EXHIBIT 13

Written Testimony of Robert A. Stern, Ph.D.

Professor of Neurology, Neurosurgery, and Anatomy & Neurobiology
Director, Clinical Core, BU Alzheimer's Disease Center
Co-Founder, Center for the Study of Traumatic Encephalopathy
Boston University School of Medicine

Before the Special Committee on Aging
United States Senate

Hearing on "State of Play: Brain Injuries and Diseases of Aging"

Wednesday, June 25, 2014

Good afternoon, Mr. Chairman, Ranking Member Collins, and distinguished Members of the Committee. It is a great honor to appear before you today for this hearing on "Brain Injuries and Diseases of Aging." My name is Dr. Robert Stern. I am a Professor of Neurology, Neurosurgery, and Anatomy & Neurobiology at Boston University School of Medicine. I am also the Director of the Clinical Core of the Boston University (BU) Alzheimer's Disease Center, one of 29 Alzheimer's research centers funded by the National Institute on Aging. In 2008, I co-founded the BU Center for the Study of Traumatic Encephalopathy (now referred to as the BU CTE Center) with Dr. Ann McKee, Dr. Robert Cantu, and Mr. Christopher Nowinski who is also testifying before you today.

For the past 25 years I have been conducting clinical neuroscience research, primarily focused on the cognitive, mood, and behavioral changes of aging, in general, and in neurodegenerative diseases, in particular. I have been on the faculties of the University of North Carolina School of Medicine, Brown Medical School, and, for the past 10 years, Boston University School of Medicine. In my role in the BU Alzheimer's Disease Center, I oversee all clinical research pertaining to Alzheimer's disease (AD), including studies aimed at the diagnosis, genetics, prevention, and treatment of this devastating disease.

Chronic Traumatic Encephalopathy (CTE)

Since 2008, my research has focused on the long-term consequences of repetitive brain trauma in athletes. In particular, I have been studying the neurodegenerative disease, chronic traumatic encephalopathy or CTE. CTE is a progressive neurodegenerative disease that can lead to dramatic changes in mood, behavior, and cognition, eventually leading to dementia. It is similar to AD but is a unique disease, easily distinguished from AD and other diseases through post-mortem neuropathological examination. CTE has been found in individuals from ages 16-98, including youth, college, and professional contact sport athletes (including football, hockey, soccer, and rugby players), military service members exposed to blast trauma and other brain injuries, and others with a history of repetitive brain trauma, such as physically abused women, developmentally disabled head bangers, and seizure disorder patients. (See **Table 1.**)

Although CTE has been known to affect boxers since the 1920s (previously referred to as "punch drunk" or dementia pugilistica), it is only recently—since CTE was diagnosed in several deceased former professional NFL players—that this disease has received greater medical and media attention. However, the scientific knowledge of CTE is in its infancy. The little that is known is based primarily on post-mortem examinations of brain tissue and interviews from the family members of the deceased athletes. What these studies have shown

is that, in some individuals, early repetitive brain trauma triggers a cascade of events in the brain leading to progressive destruction of the brain tissue. The hallmark feature of CTE is the build-up of an abnormal protein called tau (See **Figure**; based on the work of Dr. McKee), one of the abnormal proteins also seen in AD (McKee et al., 2013). These changes in the brain can begin years, or even decades, after the last brain trauma or end of athletic involvement, and can lead to memory loss, poor judgment, impulse control problems, aggression, depression, suicidality, movement problems, and, eventually, progressive dementia (See **Table 2**).

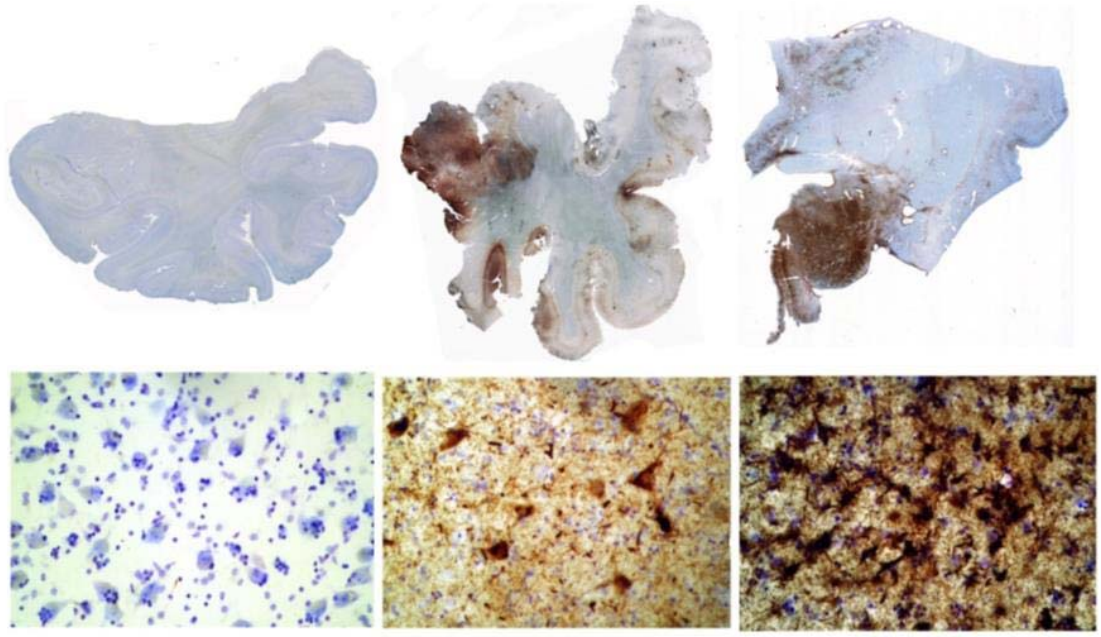

Figure of CTE Neuropathology. Left Top: Section of brain of 65 year old healthy man demonstrating no evidence of abnormal tau depositions. Left Bottom: Microscopic enhancement of same brain sample demonstrating no evidence of tau neurofibrillary tangles that would have shown up as brown from immunostain. Middle Top: Section of brain from 45 year old John Grimsley, a former NFL football player who had a five year decline in functioning (e.g., poor memory, short fuse) prior to his death from an accidental gunshot wound; brown areas are abnormal tau deposits. Middle Bottom: Microscopic enhancement of Grimsley's brain demonstrating neurofibrillary tangles. Right Top: Section of brain of 73 year old former professional boxer who died in a nursing home with clinical diagnosis of dementia pugilistica after several year decline in functioning; brown areas demonstrate widespread tau deposition. Right Bottom: microscopic enhancement of boxer's brain demonstrating widespread tau deposits

The Symptoms of CTE

Although the cognitive changes in CTE are very similar to those in AD, many individuals with CTE develop the significant changes in mood and behavior relatively early in life (Stern, et al., 2013) that can lead to significant distress for the individual with CTE as well as their family, friends, and other loved ones. These mood and behavioral impairments caused by CTE are typically misdiagnosed and attributed to routine psychiatric disorders, stress, substance abuse, or pre-existing personality traits. However, it is completely expected that the areas of the brain

damaged in CTE would lead to these problems, including depression, impulsivity, emotional lability, irritability, and behavioral dyscontrol. It is noteworthy that the much heralded "NFL Settlement" (currently in limbo while the judge examines several issues) began as a class action to address the issue of CTE in former NFL players and to provide the players and their families with appropriate compensation for the losses and distress experienced due to CTE. However, the "settlement," as it is currently written, does not provide any compensation for individuals with the mood and/or behavioral impairments so common in CTE. For example, the families of well-known former players who died of suicide and were found to have CTE post-mortem, such as Junior Seau and Dave Duerson, would not receive any benefits under the currently written settlement if they died after the acceptance of the settlement. Rather, only individuals with the memory, cognitive, and functional independence difficulties associated with Alzheimer's disease dementia would meet criteria for compensation.

Table 1. All cases of neuropathologically confirmed cases of CTE have had a history of repetitive brain trauma. CTE has been diagnosed in the following individuals:
Professional football players
College football players
High school football and other contact sport athletes
Professional soccer players
Semiprofessional soccer player
Professional rugby players
Boxers
Mixed martial art athlete
Combat military service members
Others, including a domestically abused woman, seizure disorder patients, developmentally disabled headbanger

Like other neurodegenerative diseases, CTE can only be diagnosed through post-mortem neuropathological examination of brain tissue. Dr. Ann McKee from our BU center has examined the brains of more athletes and others with repetitive brain trauma than any other neuropathologist. As part of the investigation of these post-mortem cases, I have had the great privilege and honor to interview the family members of approximately 100 deceased former athletes who were diagnosed with CTE after death by Dr. McKee and her team. From these interviews I have begun to learn about the clinical course and presentation of this disease. But, more importantly, I have learned about the tremendous pain and suffering the family members experienced while their loved one's life was destroyed by the progressive destruction of the brain. I have spoken with spouses of former professional football players who slowly lost their ability learn new information, communicate with others, dress, feed, and toilet themselves. I have interviewed

the adult children of former professional and college football and rugby players whose fathers had dramatic changes in personality, the development of aggressive and out-of-control behavior, and suicidal thoughts. And, I have spoken with the parents of young athletes in their 20's or 30's who impulsively took their own lives.

Table 2. Clinical Features of Chronic Traumatic Encephalopathy

Behavioral Features	Mood Features	Cognitive Features	Motor Features
Explosivity	Depression	Memory impairment	Ataxia
Loss of control	Hopelessness	Executive dysfunction	Dysarthria
Short fuse	Suicidality	Lack of insight	Parkinsonism
Aggression and rage	Anxiety	Perseveration	Gait Disturbance
Impulsivity	Irritability	Impaired attention	Tremor
Physical/verbal	Labile emotions	and concentration	Masked facies
violence	Apathy	Language difficulties	Rigidity
Paranoid delusions	Loss of interest	Dementia	Muscle weakness

Diagnosing CTE During Life

I also have been privileged to meet over 70 former NFL players who have come to Boston to participate in my NIH-funded research study entitled, *Diagnosing and Evaluating Traumatic Encephalopathy with Clinical Tests*, or DETECT. I hear their histories, I speak with their family members, and I listen to their fears that they have CTE or that their fellow former football players have or will get CTE. They have all witnessed firsthand the tragic downward spiral of CTE that sadly seems to have become an expected consequence of playing the game they loved. The goal of the DETECT study (which was the first grant ever funded by NIH to study CTE) is to develop objective biological tests, or biomarkers, in order to detect and diagnose CTE during life. The study involves the examination of a total of 100 former professional football players (selected based on positions played and existing clinical symptoms) and 50 same-age non-contact sport elite athletes. All research participants undergo extensive brain scans, lumbar punctures (to measure proteins in cerebrospinal fluid), electrophysiological studies, blood tests (e.g., for genetic studies and novel potential biomarkers), and in-depth neurological, neuropsychological, and psychiatric evaluations. In addition, I have recently received Department of Defense funding (with my colleague, Dr. Martha Shenton of the Brigham and Women's Hospital) and a separate grant from Avid Radiopharmaceuticals (part of Ely Lily) to examine an exciting new Positron Emission Tomography (PET) ligand (developed and owned by Avid) that is specifically designed to attach to the abnormal forms of tau protein found in CTE. Preliminary results of the DETECT study are very promising. However, it is just the first step. Future research is needed, including

longitudinal designs with much larger samples and the inclusion of newer techniques and technologies, as well as post-mortem validation of the findings during life.

To me, the ability to diagnose CTE during life is the next critical step in the study of CTE. It will lead to the ability to answer important questions about this disease, such as: How common is CTE? What are the risk factors for CTE? Can it be prevented? How can we treat it? In other words, at this point, we actually know very little about this disease (See **Table 3**). One thing we do know about CTE is that every case of post-mortem diagnosed CTE has had one thing in common: a history of repetitive brain trauma. This means that the repetitive brain trauma is a necessary factor in developing this disease. However, it is not a sufficient factor. That is, not everyone who hits their head repeatedly will develop this progressive brain disease. There are additional, as yet unknown, variables that lead to CTE, such as genetic susceptibility or specific aspects of the exposure to the brain trauma. Some have argued that brain trauma cannot possibly cause CTE, using the argument that there are many older former football players and other athletes with dramatic brain trauma history who are completely healthy. This irrational argument is analogous to those made years ago that cigarette smoking does not cause lung cancer because there are many people who smoked for decades who never develop lung cancer. An important next step in CTE research is to examine the specific additional risk factors, including genetics and exposure variables.

Subconcussive Trauma

It is important to note that CTE is not a disease restricted to former *professional* athletes. It has been found in individuals who only played their sport up through the *college* level and even just through *high school*. It has been found in warfighters who were exposed to blast trauma and other injuries. Another important issue to note is that post-mortem confirmed CTE has been found in individuals who have had no history of known or reported symptomatic concussions, but, nonetheless, were exposed to a tremendous amount of repetitive hits to the head that did not result in the symptoms of concussion. These “subconcussive” blows are quite common. It is estimated that the typical lineman in football experiences between 1000-1500 hits per season (i.e., at every snap of the ball at every play of every game and every practice), each at 20-30*g*. These hits are not just experienced by professional players. For example, a study by Broglio and colleagues (2011) found that high school football players received, on average, 652 hits to the head in excess of 15*g* of force in a single season. One player received 2,235 hits! To put this in perspective, a car going 35 mph into a brick wall experiences approximately 20*g* of force. There is now growing research evidence that even after one season, repetitive

subconcussive trauma can lead to cognitive, physiological, and structural changes to the brain. And, it appears that this exposure to repetitive subconcussive blows is associated with the development of CTE. This, perhaps, is one of the most frightening aspects of CTE. Over the past few years, there has been a tremendous increase in public awareness of *concussions* and the need to prevent and manage them. The "concussion crisis" in sports is a hot topic in the media, on playing fields, and in doctor's offices. However, when it comes to the long-term consequences of sports-related brain trauma, concussions are likely the tip of the iceberg. That is, subconcussive trauma appears to be as important or more important in the development of CTE.

Table 3. CTE Research is in its Infancy: What are the Important Questions to Address?
How common is CTE?
Is it a critical public health issue?
Above and beyond having a history of repetitive brain trauma, what are the risk factors for CTE?
Do genetics play a role in determining who gets CTE?
What types of brain trauma exposure increase risk?
Is there a certain age in childhood or adolescence when the brain is more vulnerable to brain trauma, increasing CTE risk?
How can we diagnose CTE during life?
Are there specific biomarkers that can accurately detect the abnormal tau deposition in the brain during life?
Can we distinguish between Alzheimer's disease and CTE by clinical examination?
How can we treat the symptoms of CTE effectively?
Can we modify the disease course if we intervene early?
Can CTE be prevented?
What is the biological mechanism for the development of CTE?
How does the abnormal tau move from one part of the brain to another?

Increased Funding for CTE Research

In order to tackle the complex issue of CTE, we must expand upon current approaches to conducting research in neurodegenerative disease. We must break down the traditional silos of individual research labs, research institutions, and disciplines, and begin to conduct multidisciplinary, collaborative research across research centers, bringing together the very best scientists, novel methodologies, and state-of-the-art technology. Most importantly, we must not forget that our research must focus on reducing individual human suffering and improving public health. Alas, this requires tremendous financial support. And, as you all know, current NIH funding is tragically low. The budget cuts to NIH in recent years have resulted in a tragic slowdown in the momentum of scientific discovery, and have led many scientists -- both young

investigators and older senior researchers – to leave their careers in the biomedical sciences. A recent survey by the *Chronicles of Higher Education* (Baskin & Vossen, 2014) of 11,000 senior researchers found that almost half of the respondents already abandoned an area of scientific investigation they considered key to their lab's mission. And more than three-quarters had reduced or eliminated their recruitment of graduate students and post-doctoral fellows because of reduced funding.

I want to express my deepest gratitude toward this Committee and its members for leading the recent effort to increase NIH funding of Alzheimer's disease research. However, **we must have additional funding to support research focusing on CTE and the long-term consequences of repetitive brain trauma in athletes, military service members, and other members of society**. In addition to direct federal funding, this effort will require public-private collaborative funding, such as that which supported the revolutionary Alzheimer's Disease Neuroimaging Initiative or ADNI. What might come as a surprise is that in 2012, the National Football League (NFL) donated $30 million to the Foundation for NIH to support peer-reviewed research studies on injuries affecting athletes, with brain trauma being the primary area of focus. However, that is just the beginning. We need much, much more.

In summary, many of our most cherished games in our country, such as football, hockey, and soccer, often involve repetitive blows to the head, potentially leading to a progressive brain disease with later life behavior, mood, and cognitive changes, as well as the development of dementia. We must learn as much as possible, as quickly as possible, in order to determine who may be at increased risk for CTE and other long-term consequences of the repetitive head impacts experienced by athletes at all ages, and to develop methods of preventing and treating the symptoms of CTE. I want to close by thanking the Committee for your interest in addressing this important issue and for your commitment toward improving the health and well-being of older Americans.

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EXHIBIT 14

76 of 79 Deceased NFL Players Found to Have Brain Disease

September 30, 2014, 2:57 pm ET by Jason M. Breslow

As the NFL nears an end to its long-running legal battle over concussions, new data from the nation's largest brain bank focused on traumatic brain injury has found evidence of a degenerative brain disease in 76 of the 79 former players it's examined.

The findings represent a more than twofold increase in the number of cases of chronic traumatic encephalopathy, or CTE, that have been reported by the Department of Veterans Affairs' brain repository in Bedford, Mass.

League of Denial, FRONTLINE's investigation into the NFL's concussion crisis airs tonight on many PBS stations. (Check local listings.)

Researchers there have now examined the brain tissue of 128 football players who, before their deaths, played the game professionally, semi-professionally, in college or in high school. Of that sample, 101 players, or just under 80 percent, tested positive for CTE.

To be sure, players represented in the data represent a skewed population. CTE can only be definitively identified posthumously, and many of the players who have donated their brains for research suspected that they may have had the disease while still alive. For example, former Chicago Bears star Dave Duerson committed suicide in 2011 by shooting himself in the chest, reportedly to preserve his brain for examination.

Nonetheless, Dr. Ann McKee, the director of the brain bank, believes the findings suggest a clear link between football and traumatic brain injury.

"Obviously this high percentage of living individuals is not suffering from CTE," said

McKee, a neuropathologist who directs the brain bank as part of a collaboration between the VA and Boston University's CTE Center. But "playing football, and the higher the level you play football and the longer you play football, the higher your risk."

An NFL spokesman did not respond to several requests for comment.

CTE occurs when repetitive head trauma begins to produce abnormal proteins in the brain known as "tau." The tau proteins work to essentially form tangles around the brain's blood vessels, interrupting normal functioning and eventually killing nerve cells themselves. Patients with less advanced forms of the disease can suffer from mood disorders, such as depression and bouts of rage, while those with more severe cases can experience confusion, memory loss and advanced dementia.

Among the NFL legends found to have had CTE are Duerson, Hall of Fame Pittsburgh Steelers center Mike Webster and former San Diego Chargers legend Junior Seau. On Monday, ESPN's *Outside the Lines* reported that a New York neuropathologist had discovered signs of CTE in the brain of Jovan Belcher. In 2012, the former Kansas City Chiefs linebacker shot and killed his girlfriend before driving to a Chiefs practice facility, where he committed suicide in front of team officials.

The new data from the VA/BU repository — once the "preferred" brain bank of the NFL — comes as thousands of NFL retirees and their beneficiaries approach an Oct. 14 deadline to decide whether to opt out of a proposed settlement in the class-action concussion case brought against the league by more than 4,500 former players.

The research helps address what had been a key sticking point in negotiations — the issue of prevalence. Players in the lawsuit have accused the league of concealing a link between football and brain disease. While the settlement includes no admission of wrongdoing, actuarial data filed in federal court this month showed the NFL expects nearly a third of all retired players to develop a long-term cognitive problem, such as Alzheimer's disease or dementia, as a result of football.

Under the proposed settlement, the survivors of players found to have died with CTE can qualify for a payment as high as $4 million. But some, including the family of Junior Seau, have announced plans to opt out of the settlement. Like Duerson, Seau committed suicide in 2012 by shooting himself in the chest with a .357 Magnum revolver. His family has filed a wrongful death suit against the league, arguing in part that the deal does not include adequate compensation for the descendants of former players. An attorney for the family told ESPN this month that the family was not suing "for his pain and suffering. They're suing for their own."

Others have challenged the settlement's award structure for CTE specifically, claiming it only allows for such payments if a player was diagnosed with the disease before the day that the agreement won preliminary approval in July. This detail, they say, would shut out any player who may be diagnosed in the future.

Brad Karp, an outside counsel for the league, told FRONTLINE in an e-mail that "criticism of the settlement on this ground reflects a profound misunderstanding" of the proposed agreement. "The settlement provides very substantial monetary compensation for players who suffer from the significant neurocognitive symptoms alleged to be associated with CTE and who demonstrate, through diagnostic testing, that they have moderate or severe dementia."

It remains unclear just how many players will decide to either opt out of the settlement, or choose to file a formal objection. A key test will come in November when the judge in the case holds a Fairness Hearing to consider any such challenges. Final approval would not come until sometime soon thereafter.

(AP Photo)

EXHIBIT 15

January 18, 2007

Expert Ties Ex-Player's Suicide to Brain Damage

By ALAN SCHWARZ

Since the former National Football League player Andre Waters killed himself in November, an explanation for his suicide has remained a mystery. But after examining remains of Mr. Waters's brain, a neuropathologist in Pittsburgh is claiming that Mr. Waters had sustained brain damage from playing football and he says that led to his depression and ultimate death.

The neuropathologist, Dr. Bennet Omalu of the University of Pittsburgh, a leading expert in forensic pathology, determined that Mr. Waters's brain tissue had degenerated into that of an 85-year-old man with similar characteristics as those of early-stage Alzheimer's victims. Dr. Omalu said he believed that the damage was either caused or drastically expedited by successive concussions Mr. Waters, 44, had sustained playing football.

In a telephone interview, Dr. Omalu said that brain trauma "is the significant contributory factor" to Mr. Waters's brain damage, "no matter how you look at it, distort it, bend it. It's the significant forensic factor given the global scenario."

He added that although he planned further investigation, the depression that family members recalled Mr. Waters exhibiting in his final years was almost certainly exacerbated, if not caused, by the state of his brain — and that if he had lived, within 10 or 15 years "Andre Waters would have been fully incapacitated."

Dr. Omalu's claims of Mr. Waters's brain deterioration — which have not been corroborated or reviewed — add to the mounting scientific debate over whether victims of multiple concussions, and specifically longtime N.F.L. players who may or may not know their full history of brain trauma, are at heightened risk of depression, dementia and suicide as early as midlife.

The N.F.L. declined to comment on Mr. Waters's case specifically. A member of the league's mild traumatic brain injury committee, Dr. Andrew Tucker, said that the N.F.L. was beginning a study of retired players later this year to examine the more general issue of football concussions and subsequent depression.

"The picture is not really complete until we have the opportunity to look at the same group of people over time," said Dr. Tucker, also team physician of the Baltimore Ravens.

The Waters discovery began solely on the hunch of Chris Nowinski, a former Harvard football player and professional wrestler whose repeated concussions ended his career, left him with severe migraines and depression, and compelled him to expose the effects of contact-sport brain trauma. After hearing of the suicide, Mr. Nowinski phoned Mr. Waters's sister Sandra Pinkney with a ghoulish request: to borrow the remains of her brother's brain.

The condition that Mr. Nowinski suspected might be found in Mr. Waters's brain cannot be revealed by a scan of a living person; brain tissue must be examined under a microscope. "You don't usually get brains to examine of 44-year-old ex-football players who likely had depression and who have committed suicide," Mr. Nowinski said. "It's extremely rare."

As Ms. Pinkney listened to Mr. Nowinski explain his rationale, she realized that the request was less creepy than credible. Her family wondered why Mr. Waters, a hard-hitting N.F.L. safety from 1984 to 1995 known as a generally gregarious and giving man, spiraled down to the point of killing himself.

Ms. Pinkney signed the release forms in mid-December, allowing Mr. Nowinski to have four pieces of Mr. Waters's brain shipped overnight in formaldehyde from the Hillsborough County, Fla., medical examiner's office to Dr. Omalu in Pittsburgh for examination.

He chose Dr. Omalu both for his expertise in the field of neuropathology and for his rare experience in the football industry. Because he was coincidentally situated in Pittsburgh, he had examined the brains of two former Pittsburgh Steelers players who were discovered to have had postconcussive brain dysfunction: Mike Webster, who became homeless and cognitively impaired before dying of heart failure in 2002; and Terry Long, who committed suicide in 2005.

Mr. Nowinski, a former World Wrestling Entertainment star working in Boston as a pharmaceutical consultant, and the Waters family have spent the last six weeks becoming unlikely friends and allies. Each wants to sound an alarm to athletes and their families that repeated concussions can, some 20 years after the fact, have devastating consequences if left unrecognized and untreated — a stance already taken in some scientific journals.

"The young kids need to understand; the parents need to be taught," said Kwana Pittman, 31, Mr. Waters's niece and an administrator at the water company near her home in Pahokee, Fla. "I just want there to be more teaching and for them to take the proper steps as far as treating them.

"Don't send them back out on these fields. They boost it up in their heads that, you know, 'You tough, you tough.' "

Mr. Nowinski was one of those tough kids. As an all-Ivy League defensive tackle at Harvard in the late 1990s, he sustained two concussions, though like many athletes he did not report them to his coaches because he neither understood their severity nor wanted to appear weak. As a professional wrestler he sustained four more, forcing him to retire in 2004. After he developed severe migraines and depression, he wanted to learn more about concussions and their effects.

That research resulted in a book published last year, "Head Games: Football's Concussion Crisis," in which he detailed both public misunderstanding of concussions as well as what he called "the N.F.L.'s tobacco-industry-like refusal to acknowledge the depths of the problem."

Football's machismo has long euphemized concussions as bell-ringers or dings, but what also alarmed Mr. Nowinski, 28, was that studies conducted by the N.F.L. on the effects of concussions in players "went against just about every study on sports concussions published in the last 20 years."

Studies of more than 2,500 former N.F.L. players by the Center for the Study of Retired Athletes, based at the University of North Carolina, found that cognitive impairment, Alzheimer's-like symptoms and depression rose proportionately with the number of concussions they had sustained. That information, combined with the revelations that Mr. Webster and Mr. Long suffered from mental impairment before their deaths, compelled Mr. Nowinski to promote awareness of brain trauma's latent effects.

Then, while at work on Nov. 20, he read on Sports Illustrated's Web site, si.com, that Mr. Waters had shot himself in the head in his home in Tampa, Fla., early that morning. He read appraisals that Mr. Waters, who retired in 1995 and had spent many years as an assistant coach at several small colleges — including Fort Valley (Ga.) State last fall — had been an outwardly happy person despite his disappointment at not landing a coaching job in the N.F.L.

Remembering Mr. Waters's reputation as one of football's hardest-hitting defensive players while with the Philadelphia Eagles, and knowing what he did about the psychological effects of concussions, Mr. Nowinski searched the Internet for any such history Mr. Waters might have had.

It was striking, Mr. Nowinski said. Asked in 1994 by The Philadelphia Inquirer to count his career concussions, Mr. Waters replied, "I think I lost count at 15." He later added: "I just wouldn't say anything. I'd sniff some smelling salts, then go back in there."

Mr. Nowinski also found a note in the Inquirer in 1991 about how Mr. Waters had been hospitalized after sustaining a concussion in a game against Tampa Bay and experiencing a seizure-like episode on the team plane that was later diagnosed as body cramps; Mr. Waters played the next week.

Because of Dr. Omalu's experience on the Webster and Long cases, Mr. Nowinski wanted him to examine the remaining pieces of Mr. Waters's brain — each about the size of a small plum — for signs of chronic traumatic encephalopathy, the tangled threads of abnormal proteins that have been found to cause cognitive and intellectual dysfunction, including major depression. Mr. Nowinski tracked down the local medical examiner responsible for Mr. Waters's body, Dr. Leszek Chrostowski, who via e-mail initially doubted that concussions and suicide could be related.

Mr. Nowinski forwarded the Center for the Study of Retired Athletes' studies and other materials, and after several weeks of back-and-forth was told that the few remains of Mr. Waters's brain — which because Waters had committed suicide had been preserved for procedural forensic purposes before the burial — would be released only with his family's permission.

Mr. Nowinski said his call to Mr. Waters's mother, Willie Ola Perry, was "the most difficult cold-call I've ever been a part of."

When Mr. Waters's sister Tracy Lane returned Mr. Nowinski's message, he told her, "I think there's an outside chance that there might be more to the story."

"I explained who I was, what I've been doing, and told her about Terry Long — and said there's a long shot that this is a similar case," Mr. Nowinski said.

Ms. Lane and another sister, Sandra Pinkney, researched Mr. Nowinski's background, his expertise and

experience with concussions, and decided to trust his desire to help other players.

“I said, ‘You know what, the only reason I’m doing this is because you were a victim,’ ” said Ms. Pittman, Mr. Waters’s niece. “I feel like when people have been through things that similar or same as another person, they can relate and their heart is in it more. Because they can feel what this other person is going through.”

Three weeks later, on Jan. 4, Dr. Omalu’s tests revealed that Mr. Waters’s brain resembled that of an octogenarian Alzheimer’s patient. Nowinski said he felt a dual rush — of sadness and success.

“Certainly a very large part of me was saddened,” he said. “I can only imagine with that much physical damage in your brain, what that must have felt like for him.” Then again, Mr. Nowinski does have an inkling.

“I have maybe a small window of understanding that other people don’t, just because I have certain bad days that when I know my brain doesn’t work as well as it does on other days — and I can tell,” he said. “But I know and I understand, and that helps me deal with it because I know it’ll probably be fine tomorrow. I don’t know what I would do if I didn’t know.”

When informed of the Waters findings, Dr. Julian Bailes, medical director for the Center for the Study of Retired Athletes and the chairman of the department of neurosurgery at West Virginia University, said, “Unfortunately, I’m not shocked.”

In a survey of more than 2,500 former players, the Center for the Study of Retired Athletes found that those who had sustained three or more concussions were three times more likely to experience “significant memory problems” and five times more likely to develop earlier onset of Alzheimer’s disease. A new study, to be published later this year, finds a similar relationship between sustaining three or more concussions and clinical depression.

Dr. Bailes and other experts have claimed the N.F.L. has minimized the risks of brain trauma at all levels of football by allowing players who sustain a concussion in games — like Jets wide receiver Laveranues Coles last month — to return to play the same day if they appear to have recovered. The N.F.L.’s mild traumatic brain injury committee has published several papers in the journal Neurosurgery defending that practice and unveiling its research that players from 1996 through 2001 who sustained three or more concussions “did not demonstrate evidence of neurocognitive decline.”

A primary criticism of these papers has been that the N.F.L. studied only active players, not retirees who had reached middle age. Dr. Mark Lovell, another member of the league’s committee, responded that a study using long-term testing and monitoring of the same players from relative youth to adulthood was necessary to properly assess the issue.

“We want to apply scientific rigor to this issue to make sure that we’re really getting at the underlying cause of what’s happening,” Dr. Lovell said. “You cannot tell that from a survey.”

Dr. Kevin Guskiewicz is the director of the Center for the Study of Retired Athletes and a member of U.N.C.’s department of exercise and sport science. He defended his organization’s research: “I think that

some of the folks within the N.F.L. have chosen to ignore some of these earlier findings, and I question how many more, be it a large study like ours, or single-case studies like Terry Long, Mike Webster, whomever it may be, it will take for them to wake up."

The N.F.L. players' association, which helps finance the Center for the Study of Retired Athletes, did not return a phone call seeking comment on the Waters findings. But Merril Hoge, a former Pittsburgh Steelers running back and current ESPN analyst whose career was ended by severe concussions, said that all players — from retirees to active players to those in youth leagues — need better education about the risks of brain trauma.

"We understand, as players, the ramifications and dangers of paralysis for one reason — we see a person in a wheelchair and can identify with that visually," said Mr. Hoge, 41, who played on the Steelers with Mr. Webster and Mr. Long. "When somebody has had brain trauma to a level that they do not function normally, we don't see that. We don't witness a person walking around lost or drooling or confused, because they can't be out in society."

Clearly, not all players with long concussion histories have met gruesome ends — the star quarterbacks Steve Young and Troy Aikman, for example, were forced to retire early after successive brain trauma and have not publicly acknowledged any problems. But the experiences of Mr. Hoge, Al Toon (the former Jets receiver who considered suicide after repeated concussions) and the unnamed retired players interviewed by the Center for the Study of Retired Athletes suggest that others have not sidestepped a collision with football's less glorified legacy.

"We always had the question of why — why did my uncle do this?" said Ms. Pittman, Mr. Waters's niece. "Chris told me to trust him with all these tests on the brain, that we could find out more and help other people. And he kept his word."